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THE CANADIAN INTERN BOARD

ROBERT B. ANDREW

The Canadian Intern Board was founded in 1939 as an outgrowth of the Canadian Association of Medical Students and Interns. It was created with the intention of simplifying the appointments of interns to Canadian hospitals and at the same time assuring maximum satisfaction to hospitals and interns concerned. Its present officers are

Honorary Chairman.....H. S. Little, M.D.
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In addition there is an advisory committee representing the various medical centers throughout the country.

The mechanism of the board is best explained in a quotation from "Procedure for the Appointment of Interns—1945-46".

"1. Each medical student will apply for an internship to the administrator or the intern committee of the hospitals in one of which he desires to intern.

All applications must be made to the hospitals before "a specific date".

2. The medical student will enter on a Form A (supplied by the Canadian Intern Board) the names of the hospitals to which he has applied for an internship in the order of his preference and send the form to the Canadian Intern Board before "a specific date".

3. The hospital administrator or Intern Committee will rank the applicants in the following two groups, from an investigation of the applications, personal interviews, hospital examinations, letters of recommendation or any other data that may be available:

Group 1—Those that the hospital definitely desires as interns;

Group 2—Those that the hospital will appoint should any applicants in Group 1 be eliminated, i.e., alternates or second choices, listed in order of preference.

The hospital will enter the names of the applicants in these two groups on Form B (to be supplied by the Canadian Intern Board) and send the form to this Board on or before "a specific date".

4. The Canadian Intern Board will examine Forms A and B, endorse each hospital list of applicants in Group 1, if no other hospital has placed the same applicant in Group 1. In the case of those applicants which have been placed in Group 1 by two or more hospitals, the Board will endorse the hospital list in which the applicant has placed the hospital highest in order of his preference on Form A, and will delete his name from all other hospital lists, thus creating vacancies for alternates in these hospitals.

The Board will send to each hospital the list of interns as it has been revised placed in Group 2 on Form B submitted by the respective hospitals, and the same method followed as above. By this arrangement the hospitals will still control the filling of any gaps on their selected list of interns.

The Board will continue this process of dovetailing the hospitals' choice of applicants and the students' choice of hospitals until the lists of choice have been exhausted.

The Board will send to each hospital the list of interns as it has been revised by the above procedure by "a specific date", and the hospital will notify the applicants of their appointment as intern as soon as possible. This can be done only if the hospitals co-operate by making prompt reply. On the receipt of his notification of appointment, each applicant must notify the hospital of his acceptance immediately.

It is quite likely that vacancies in some of the hospitals will not be filled and that some of the students may not be placed. In order to bring these together, the Board will send to those hospitals with unfilled positions the names of the students without appointments and will send these students who are not appointed to any of the hospitals, a list of hospitals in which vacancies are still present. Students

may then make application to these hospitals and the hospitals could communicate directly with the students.

Result

By this scheme, each hospital would secure the interns considered by it most desirable, and each medical student would receive the best appointment open to him in accordance with his expressed preference. Experience has already proved that a higher percentage of candidates applying through the Canadian Intern Board has received desired appointments than was ever formerly possible through individual application alone.

Policy

The C.I.B. will make every effort to give complete satisfaction and to fulfil the desires of the hospitals and of interns. In the operation of this plan the Board will adhere rigidly to the rules of procedure as agreed upon by hospitals and medical students. For the successful operation of this Board every student is urged to accept the appointment created for him through the Board. Any unforeseen problems arising under the above plan will be dealt with in co-operation with the Advisory Committee.

Internships affected by the Plan

For the present, undergraduate internships which are arranged by the universities, senior internships, residences, research positions and similar appointments are not included in this plan, as appointments to these do not present the same problems as do the appointments of graduates to junior internships. With continued successful operation of the Board it is hoped that the scope of this plan may be eventually increased so as to aid in the allocation of senior appointments."

This system has been functioning efficiently in Toronto during the past few years. True, it has its apparent faults. For instance, in the last graduating class at Toronto, several students were left without appointments when the lists were published in December. This was due to several extenuating circumstances probably the most effective being the return of ex-servicemen to Senior Internships in many of the larger hospitals. In fairness to all, this necessitated a reduction in the number of junior appointments at these institutions. Several of those without appointments were able to secure, as outlined above, lists of hospitals with internships still open. One must realize that had no such system existed, confusion would have run riot.

At this point let it be stressed that a student should not apply to one, two, or even three hospitals and lean back confident that he is "set" for the next year of his medical life. Application should be made to a sufficient number of hospitals to bar any chance of last minute hurried letters and telegrams on finding oneself without an appointment.

As yet the board has not been functioning at all medical schools or hospitals. It is hoped that in time the Board may be extended to include the entire country.

This is desirable for it provides a strong organization which can act as a stabilizing force in intern appointments. For instance, certain hospitals have complained that students have contracted, perhaps only verbally, to intern in their institution, only to renege, when finding a more desirable post elsewhere. Further, hospitals have been known to withdraw an appointment already guaranteed to one when another with better qualifications has appeared. This in both instances is unfair for it upsets the plans of all concerned. A strong board, representative of both hospitals and students, could intervene to minimize such disrupting occurrences.

In conclusion let it be pointed out that the Intern Board serves as a connecting link, a directing force between hospitals and students and is in no way influential in specific intern appointments. Its existence is in every way a distinct advantage over the old cut-throat system of making and securing intern appointments.

INFANTILE DIARRHOEA

D. C. ROSS, M.D.

Until very recent times the cradle has provided some of the most fertile soil for disease, and there is none that has ravished it more cruelly than infantile diarrhœa, especially the severe form which was known to our grandmothers as "summer complaint". This disease, in spite of its mild sounding name, was notorious for the rapidity with which it could produce death, leaving an emaciated, wizened, little corpse, where a few days before there had been a healthy, chubby infant. In its presence the doctor was as helpless as the distracted mother. He treated empirically and vigorously, and often with unfavourable results.

The therapeutic agents which enjoyed the most widespread popularity were calomel, castor oil and whiskey. It was believed that the diarrhœa was due, either to an accumulation of indigestible curds or to the irritation of decomposed food, and so the calomel and castor oil were given in an effort to throw off the irritating masses contained in the bowel, to relieve the engorged mucous membrane and to start secretion, and the whiskey was administered as a stimulant. This basic treatment was supplemented by such procedures as lancing the gums if they seemed at all tender, bathing in hot mustard water, wrapping in flannel, and sending the child away to breathe cool air close to water, as it had been noticed that the disease was most prevalent in the hot, dry, dusty atmosphere of the cities. One authority around 1880 advises that where the infant is breast fed, the doctor should in addition to the above therapy administer "A good dose of castor oil" to the mother, as he feels that there is probably something in her milk due to an indiscretion of diet, which disagrees with the child.

There was little progress made in treatment until it was appreciated that children suffering from diarrhœa actually died of dehydration and acidosis, and as these children were usually vomiting, an effective method had to be developed for replacing fluids. In 1910 intravenous medication became practical as a result of the work of Ehrlich, and since that time it has been possible to evolve a rational method of treatment in this disease.

Although the etiology of infantile diarrhœa is obscure, it is necessary for purposes of discussion to have a classification, and the following one which is used at the Hospital for Sick Children, Toronto, is offered as one which is acceptable in the light of present knowledge. Under this scheme, infantile diarrhœa is divided into four main groups as follows:

Group I —Infectious diarrhœa

Group II —Metabolic diarrhœa

(a) Acute—Fermentative diarrhœa

(b) Chronic—Cœliac disease

Fibrocystic disease of the pancreas

Group III—Infectious intoxication

From the wards of the Hospital for Sick Children under the direction of Alan Brown, M.D., F.R.C.P. (Can.)

Group IV—Intestinal intoxication

Group I— Infectious Diarrhoea. This group is made up of those cases of diarrhœa produced by specific organisms, such as the dysentery and paradysentery groups, and the salomella group, e.g., B. Typhi Murium. The only way that this disease can be suspected clinically is by the history of mucus or pus and blood in the stools, as this is an unusual finding in the non-specific group of diarrhœa. The final diagnosis is, however, bacteriological.

Group II—Metabolic diarrhoea

(a) Acute—Fermentative diarrhœa. Fermentative diarrhœa results from the fermentative, acid-producing action of the intestinal organisms of the large intestine on carbohydrate. These acids act as direct chemical irritants upon the mucous membrane causing increased intestinal peristalsis and frequent loose stools. As some form of carbohydrates are much less readily fermented than others, and as some are absorbed more quickly than others and therefore do not reach the large intestine where this action occurs, the amount of irritant acid produced varies with the form in which the carbohydrate is administered. Lactose is the most easily fermented and also the most slowly absorbed of all the sugars used in infant feeding, and so is the most laxative. On the other hand, the flours do not ferment easily and so are the least laxative form of administering carbohydrate. Between these two extremes lie the three other sugars commonly used in infant feeding, which in order of their fermentability are corn syrup, granulated sugar and dextri maltose. In the presence of this type of diarrhœa it is therefore necessary to change to one of the less laxative forms of carbohydrate, and where the diarrhœa is severe, to reduce or omit the carbohydrate and supply the caloric requirements by adding additional protein, usually casein, to the feeding. From protein little if any acid is produced by bacterial decomposition, so that the stools become alkaline in reaction and constipated in type.

(b) Chronic. The chronic, metabolic type of diarrhœa is manifested in the cœliac syndrome and fibrocystic disease of the pancreas, two disease entities which are characterized by large, foul-smelling stools, porridge-like in consistency.

Group III. Infectious intoxication. In these children the diarrhœa is part of a general intoxication accompanying an acute parenteral infection, which is usually respiratory but may be located anywhere in the body. In some the diarrhœa may be absent even though they show other signs of toxicity such as paleness, drowsiness, convulsions, and in severe cases peripheral vascular collapse. In just what manner the parenteral infection acts to produce diarrhœa is not known, but presumably it in some way provides a sufficient stress to upset the digestive processes which in infants are never very stable.

Group IV. Intestinal intoxication. Intestinal intoxication presents a picture similar to group III, except that there is no evidence of parenteral infection. In the full-blow case the temperature is elevated, the infant is a pale gray colour, often likened to that of wet ashes, is shocked, drowsy and is usually quite obviously

dehydrated, with sunken fontanelle, poor tissue turgor, dry skin, dull sunken eyes, a wrinkled, pinched face, an extremely emaciated body, and a soft, scaphoid, doughy abdomen. The transformation from a normal well infant to the picture described can occur in an amazingly short time, and it is not at all uncommon for the parents to say that the child was perfectly well up to two or three days before.

It is thought that the disease is due to enteral infection, but as specific pathogenic organisms are frequently not isolated from the stools, it has been difficult to prove this conclusively and a great deal of investigation has been carried out concerning the possibility of normal colon inhabitants becoming pathogenic under certain conditions. Many investigators (Bessau and Bossert, 1919; Moro, 1929; Deak, 1933; Blacklock et al, 1939) have shown that in this disease, with or without clinically demonstrable parenteral infection, the upper small intestine which is normally sterile or contains only a few enterococci may harbour large numbers of coliform organisms which have made their way upwards from below. Blacklock et al, 1937, found that in older children with parenteral infections there was also an increase in the coliform organisms, in the upper small intestine, but the change was not so marked as in infants. The coliform organisms which gain the upper small intestine were thought to take on a pathogenic role in their new environment and have been shown by Goldschmidt 1931 to produce on culture outside the body toxic substances which in small doses irritate the intestine and in larger doses actually paralyze it. Brown A. et al, 1933, however, in a very careful investigation of the stools and serology of patients with diarrhœa symptoms found that specific organisms of the colon paratyphoid paradysentery group were much more frequently the cause than commonly supposed, and they concluded from this investigation that acute intestinal intoxication, infectious diarrhœa, dysentery, and summer diarrhœa were synonyms indicating one condition—a gastrointestinal infection with a species of the colon paratyphoid dysentery group of bacilli, and that certain diagnosed fermentative diarrhœa also belonged to this group.

In some of the cases where no specific organisms are found it is difficult to rule out an associated parenteral infection, possibly virus in origin, sufficiently mild to pass undetected but still capable of producing an intoxication. Such cases would rightly belong to group III, the infectious intoxication group.

In contrast to the doubt in which the etiology of this disease has been shrouded there stands out a very pertinent and important fact which has been confirmed over and over again. Infantile diarrhœa is infrequent in breast fed infants, and this is a truism which has been recognized for many years and is one of the strongest arguments in favour of breast feeding, especially amongst the lower income groups where facilities are inadequate and proper technique in the preparation of feedings is apt to be neglected.

Infantile diarrhœa is a disease of the summer months, the season when flies are plentiful, when the temperature is conducive to bacterial growth, and when

the infant and his stools are most likely to be exposed. Prophylactic measures therefore should include:

(1) Breast feeding wherever possible.

(2) Protection of the infant, his clothes, and food, from flies.

(3) Scrupulous care in the preparation of artificial feedings.

(4) The use of evaporated, tinned milk in artificial feedings, as it has been superheated and is therefore sterile at the time the tin is opened, is more digestible than ordinary whole milk, and in addition frees the mother from dependence on the local milk supply, especially important in rural areas and summer places.

(5) When made up, the day's feedings should be stored in a refrigerator until time for use.

The treatment of infantile diarrhœa as carried out at the Hospital for Sick Children divides itself into the treatment of (a) the infection; (b) the dehydration; (c) the toxicity and shock.

The treatment of the infection is chemotherapy with sulphadiazine in the non-specific, specific and parenteral infections, and in addition penicillin should be used in those cases where there is a parenteral infection caused by a penicillin sensitive organism. Abscesses should be drained, bulging ear drums opened, infected mastoids treated by mastoidectomy, and every effort made to rid the child of infection.

The dehydration should be treated by fluids both by mouth and parenterally. 15% sugar solution and citrated orange juice are offered every half hour for 2 to 3 days until the dehydration and toxicity have improved, and then the child is started on a dilute milk formula which is gradually strengthened as the improvement continues. Very small or premature infants however are kept on milk formulæ throughout, usually a mixture of two-thirds breast milk and one-third protein milk, or lactic acid milk, as they do not tolerate being deprived of food. Continuous intravenous therapy is started on admission by cutting down on and inserting a gold needle into one of the veins of the extremities. The intravenous fluid used is a hypotonic solution of two-thirds 5% glucose in distilled water and one-third normal saline, and this mixture is permitted to run at the rate of 15 cc. per lb. per 12 hours. Occasionally the infant is given subcutaneous injections of the same mixture, but the handling and trauma involved in the procedure are sufficient to make the intravenous route the one of choice.

Shock and toxicity are treated by small transfusions of whole blood or plasma usually repeated once or twice.

In addition, small doses of paregoric and bismuth are given to control the

hypermobility of the intestine. This is administered in the form of a mixture made up as follows:

Tinct. of camph. co. min. ii to vi,
depending on size of child.
Bis. salicylate grs. ii
never more, regardless of size of patient.
Mistura creta ad drs. i

A convenient way of ordering this is drs. i after each loose stool, up to a maximum of five doses in any twelve hours. In this way the amount given is proportional to the severity of the symptoms, but the patient should be watched carefully as the bismuth salicylate may aggravate the acidosis. If bismuth is being ordered in the home, it is well to warn the mother that it will make the stools black.

Summary:

1. The subject of diarrhœa in infancy has been reviewed, following the classification used at the Hospital for Sick Children, and taught in the Department of Pædiatrics, University of Toronto.

2. The methods of prophylaxis have been outlined and the importance of encouraging and educating mothers to breast feed their infants as a means of preventing this disease has been stressed.

3. The treatment of severe diarrhœa accompanied by dehydration, toxicity and infection, has been described as it is carried out on the wards of the Hospital for Sick Children, Toronto.

The author wishes to express his appreciation and indebtedness to Dr. J. H. Ebbs for advice and guidance in the preparation of this paper.

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THE USE OF PITUITARY EXTRACT IN OBSTETRICS

OSCAR KOFMAN '46

I. Introduction

Sometimes that which the sciences have given to man falls into unsafe hands. The injudicious use in obstetrics of the extract of the posterior pituitary gland is an example of one of these indictments. Rupture of the uterus, laceration of the cervix and dead babies follow in the train of its use all too often—and yet it is still widely used indiscriminately.

II History

Pituitary extract was not introduced into obstetrics until 1909 by Blair-Bell and Hofbauer. In 1915, only a few years after Blair-Bell's pioneer paper, the enthusiasm which greeted the advent of this new agent was shown by an advertisement of a large pharmaceutical concern: "Throw away your forceps and use pituitrin". However, cases of uterine rupture soon began to appear in the literature and the early popularity gave way to a conservative attitude in many obstetrical centres; but even to the present day many have persisted in certain routine liberal uses of pituitary extract.

III Physiology and Chemical Composition

This extract of the posterior lobe of the pituitary gland contains two known parts—one, an oxytoxic portion called pitocin, and a fraction which elevates blood pressure and stimulates peristalsis, known as pitressin. However, the separation of these two fractions is not yet completed. The exact chemical composition of pituitary extract is not known.

IV Standardization

This substance is standarized in Vœgtlin units. One unit corresponds to the extract of 0.5 mgms of a standard powder of the posterior lobe of the pituitary gland, conserved under the care of the Section of Hygiene of the League of Nations and a few other laboratories of pharmacology. Physicians should specify the dose of pituitary extract in terms of units in preference to ampules or minims. Two Vœgtlin units are considered to be equivalent to three minims or 0.2 cc. of extract.

V Preparation

Many preparations of pituitary extract are on the market and although they are supposed to be standardized in Vœgtlin units, they vary not a little in strength, and what is equally important, some of them contain an appreciable amount of protein which may cause anaphylaxis. Further, combinations of pituitary extract with hormones or drugs—proprietaries under fantastic names—have enjoyed world wide recommendations and use. It is claimed that these preparations are safer than straight pituitary extract solution, that they have virtues of their own which not

only reduce the dangers of the oxytoxic, but also favorably influence labor, and obstetricians are importuned to administer these questionable drugs to all women for the purpose of hurrying delivery. According to De Lee (1940), no physiological, experimental or clinical evidence has yet been presented to prove that thymophysin, pituthymin, thytuitary, and the like are any more than diluted solutions of pituitary extract or the drug in another guise. But more recently, based on the principle that if the hormone of the posterior lobe of the pituitary was combined with some other substance so that it would be liberated very slowly, hence minimizing or eliminating the danger involved in its use, the solution of Posterior Pituitary Sulfonate has been introduced. The oxytoxic content of this substance is equal to that of pituitary extract and it is liberated more slowly. The subcutaneous or intramuscular injection in pregnant women at term, of Posterior Pituitary Sulfonate differs from the usual solutions of pituitary hormone in that the latent period is longer and there is little if any likelihood of tetanic contractions of the uterus and the duration of action is longer. However, the results of the use of this substance have not even yet been sufficiently ascertained and the fact still remains that the increasing demand among women for "streamlined labor" is unhealthy in its results and must be subdued.

VI Dangers and Complications

On considering the dangers resulting from the injection of posterior pituitary extract, we see that most come from the *increase in violence of the uterine contractions;* but other dangers are also present. These include the following:-

(1) SHOCK, which may leave permanent after-effects in the brain. Shock acts like asphyxia.

(2) HORMONAL IMBALANCE—hence must be avoided in dysthyroidism, diabetes, ergometrine being used instead.

(3) CONVULSIONS in cases of threatened eclampsia. Some reports of post partem eclampsia without premonitory symptoms with the use of pituitary extract are also found.

(4) HEART DISEASE—danger due to the increase tonus of the arterioles of the coronary system (caused by the pressor principle). Here, too, ergometrine is better.

(5) ANAPHYLAXIS is not rare. It shows itself as severe urticaria, violent pruritus, local or general, edema of the face, eyes, glottis or lungs—even severe shock.

The greatest dangers come out of the increase in the intensity and durations of the contractions of the uterus. These may reach a violence that will rupture the organ if the resistance to the advance of the projectile (the baby) is more than the strength of the exploding chamber (the uterus). Many cases of pituitary rupture of the uterus are known, and still more have not been reported. Ruptures have been known to occur even from small doses of pituitary extract—doses usually considered safe. Pituitary extract has been seen to blast the head through the

cervix and perineum, tearing both extensively. Forces may be set into motion which cannot be controlled.

Claims have been made of combinations of pituitary extract which will contract the fundus while they relax the lower uterine segment and cervix—but there has been no scientific proof of this. The more rapid dilatation of the cervix is due to the drug strengthening the contractions which force the head, with or without the bag of waters, through the parts, thus canalizing the cervix quicker. Hence we can readily understand how such violence will destroy the whole internal pelvic architecture which supports the pelvic organs in a manner so admirably adapted to bear the stresses and strains of the maternal functions for which they are intended. Even normal labor puts this well balanced muscular fascial and connective tissue framework to a severe test. The action of pituitary extract is ruthless in destroying it and the portals of entry are thrown wide open for the entry of infection.

Pituitary extract can cause such violent labor pains, combined with powerful bearing down effects, that the sudden over exertion has caused cardiac death. Uteri stimulated by pituitary extract, not seldom suffer secondary atony with clot retention, thrombosis and embolism.

As for babies, pituitary extract is of equal if not greater danger. The moulding of the head is a result of the mechanical forces of the uterus and its safety for the fœtus is only partly guaranteed by the slowness of its execution. Strongly moulded heads, from the use of pituitary extract, often show evidence of brain distortions and intracranial hæmorrhage. The brain is a very tender organ and it bears the insults of the forces of labor poorly and therefore one should not augment the intensity of these forces without very good reason.

In addition to this, pituitary extract, by causing long, violent tetany of the uterus, causes asphyxia with slowing of the fœtal heart through stoppage of the uteroplacental circulation.

The harmful action of pituitary extract in normal labor is thus clear. Pituitary extract (1) strengthens the uterine power and prolongs its action;

(2) shortens the diastolic rest periods;

(3) increases the intra-uterine tension by raising the tonus of the muscle.

In short pituitary extract makes normal pains pathologic and exaggerates all their effects on both mother and baby.

Hence we see that the physician who administers pituitary extract to accentuate pain and hasten delivery is accepting a grave responsibility. It should not be used in normal labor other than in the third stage, yet in many centres it is used most widely in this condition.

VII Medico-Legal Aspect

The medico-legal aspect of the use of pituitary extract to hurry normal labor and resulting in cerebral hæmorrhage with a spastic idiot, is worth noting. No professional standard anywhere or at any time could justify a physician in doing

anything that would jeopardize the life and well-being of mother or infant solely for the physician's convenience.

VIII Controversy—In Normal Labor

Murphy of Philadelphia, in spite of the evidence recorded above, insists on the fact that the danger which results from the improper use of pituitary extract is more a problem of dosage than a question of which patient should receive it. He claims that all the dangers of the drug are the result of the use of doses far in excess of what should have been employed. He does not believe that pituitary extract should be condemned in normal labor but the dose should be in the neighborhood of 1 or 2 minims (2/3 - 1 1/2 units).

However, in spite of this dispute, pituitary extract is one of the great blessings that science has conferred on women, and the profession is now well agreed in the majority of its scientific indications.

IX Scientific Indications

A. *During Pregnancy*:

(1) Pituitary extract causes the relaxed ureter to expell its contents and thus aid in the cure of pyelitis. Five unit doses are required here and may be used during early pregnancy and in the puerperium.

(2) In case of abortion in progress, after an appendectomy, pituitary extract may be used for the double purpose of emptying the uterus and overcoming ileus.

(3) In cases of septic abortion or abortion actually in progress, to hasten the process and decrease hæmorrhage.

(4) When currettage is done (or incomplete abortion) pituitary extract injected deeply into the cervical wall will stiffen the uterus, separate the secundines and give the curet a harder and safer surface to work on. It will also reduce the loss of blood.

B. *During Labor:*

(1) It is safe to induce labor with pituitary extract only if proper precautions are observed. Hofbauer's nasal method was found by De Lee to be dangerous. Some women react to even small doses of the oxytoxic with too strong pains, no matter how it is given. To induce labor, the following routine should be followed:

6 a.m.	castor oil, 2 oz.
7 a.m.	hot soap suds enema
8 a.m.	5 - 10 gr. quinine sulphate
9 a.m.	repeat quinine
12 a.m.	repeat quinine
2 p.m.	¼ cc. pituitary extract (2½ units)
2:30 p.m.	½ cc. pituitary extract every ½ hr. x 6 or till labor pains have begun.

During Labor Itself:

(1) Pituitary extract should not be used in normal labour; however, pituitary

extract has a limited use in the first and second stages of labor complicated by primary inertia—which is an actual deficiency in the uteromusculonervous mechanism. Considerable controversy exists in regard to indications in uterine inertia.

Schumann states as follows: "if the birth canal is clear for the passage of the foetus which lies in a normal presentation and position, then the uterus may be stimulated even though the cervix is not fully dilated and effaced. The only drug here is pituitrin (pituitary extract)".

On the other hand Davis states: "rarely is it advisable to give pituitary extract for uterine inertia. If this is done, dosage should be limited to 1 - 2 minims subcutaneously. (2/3 - 1½ units). However, Sharkey states that the use of pituitary extract in uterine inertia should be limited to cases of true primary inertia, that is actual weakness of the contractile muscle or of the uterus muscular nervous mechanism—though the real cause of the difficulty is still unknown. (Various contraindications of use of pituitary extract in uterine inertia are listed further on).

In regard to secondary inertia, Sharkey makes the following statement: "when the uterine musculature is exhaused after many hours of hard labor, pituitary extract is not to be used. Sedatives, rest, nutrition, to prepare the patient for operative delivery or further test of labor is advisable."

(2) When the second stage of labor is unduly prolonged because of the failure of weak uterine contractions to expel the fœtus, presentation being normal and rotation completed, then it is wise to terminate labor with low forceps. In home deliveries, because of the lack of facilities, this is not always possible, therefore under these conditions, pituitary extract may be used instead of forceps, preferably in multipara and in doses of one or two minims (2/3 to 1½ units). In fact, this is the only instance in which the American Committee on Maternal Welfare recommends the use of pituitary extract in the first or second stage of labor.

(3) In cases of abruptio placentæ, it is permissible to give pituitary extract, with precautions, to hurry labor and increase uterine tonus, but never in placenta prævia until the baby is delivered.

(4) The Third Stage of Labor—should pituitary extract be used as a routine in the third stage of labor? Such practice is widespread but the reports of the results are conflicting. Some state that it shortens the third stage, reduces loss of blood, favors the expulsion of the placenta, prevents postpartem haemorrhage and favors involution, which minimizes the risks of infection. Others deny these actions. On the other hand, several placental incarcerations have resulted from the use of the oxytoxic drug. However by most authorities the routine use of pituitary extract (½ to 1 cc.: 5 - 10 units) intramuscularly immediately after the baby is born, is considered valuable for reducing blood loss and shortening the period for placental delivery.

(5) Pituitary extract finds a great field of usefulness in cases of postpartem haemorrhage. In such cases, the drug may be given before or after the placenta has been expelled. If bleeding is still profuse after the uterus contracts, it is almost

certainly due to lacerations. Pituitary extract has little effect on lacerations. In cases of haemorrhage, 10 units should be given intramuscularly into the Deltoid or Gluteus Maximus or injected directly into the uterine muscle.

C. *In the Puerperium:*

Pituitary extract has yielded to ergometrine in nearly all cases in which an oxytoxic is needed during the puerperium. Amongst such cases are expulsion of a piece of placenta or a clot that remains in the uterus, or in late haemorrhage of a hormone nature.

D. *Other Indications:*

Pituitary extract is also used in obstetrics as follows:

(1) In some cases of hydatidiform mole in which the patient is bleeding, 5 units given intramuscularly may cause an expulsion of a part of the mole and thereby permit a correct diagnosis without invasion of the uterus.

(2) During Cæsarian sections, immediately after the baby is born, ten units of pituitary extract should be injected directly into the uterine muscle. This favors rapid separation of the placenta and reduces the amount of bleeding from the uterine incision and the placental tissues.

X CONTRAINDICATIONS:

A. *General:*

In general we may state that pituitary extract should not be used when uterine inertia is due to the following mechanical causes:

(1) generally contracted pelvis.
(2) abnormal position of the fœtus.
(3) tumors of the uterine wall or other tumors in or near the uterus blocking the birth canal.
(4) congenital anomalies such as bicornate uterus or infantile uterus.
(5) abnormal positions of the uterus, as retroflexion, or anteflexion with pendulous abdomen.
(6) great multiparity causing thinning of the uterine wall with the danger of spontaneous ruptures of the uterus.
(7) overdistention of the uterus as in polyhydramnios, and multiple pregnancies.
(8) scars in the uterus from previous operations or disease of the uterine wall.
(9) dystocia dystrophia syndrome.
(10) rigid cervix undilatable.

A critical examination of the causes just given will show that operative intervention will often be necessary to accomplish delivery and that in the treatment of uterine inertia the underlying cause is important.

B. *During Complications*:

In considering the complications of pregnancy we find the following contraindications to the use of pituitary extract:

(1) Toxemias of pregnancy.—In toxemic patients pituitary extract should not be used because of its potential dangers. It has been shown that the blood pressure may rise an average of 51 mm. of mercury after the misdirected use of the drug in toxemias and that it may reduce diuresis and cause convulsions or pulmonary edema if the patient is edematous.

(2) Abruptio Placentæ—Pituitary extract in conjunction with the abdominal binder has been recommended in the treatment of abruptia placentæ. This treatment should be administered only by a skilled obstetrician with sound judgment.

(3) Placenta Prævia—If intrapartum hæmorrhage is due to low implantation of the placenta, pituitary extract will not control the bleeding and the drug should not be given for this purpose.

(4) Heart disease—Pituitary extract should not be used in serious heart disease because of the danger of reactions and the added strain placed on the heart should the labor become severe following injection of the drug.

XI Dosage and Administration:

The dose of pituitary extract should always be small and should not exceed three minims (0.18 cc.) subcutaneous, while the child is in the uterus. If one prescribes unit dosage, the standard preparations are marketed with ten international units per cubic centimeter or two-thirds unit per minim. Murphy's studies on the pregnant uterus at term with the Lorand tocograph indicate that, if tetanic contractions are to be avoided, the dose has to be kept below one and one-half minims (0.09 cc.), and that there should be an interval of thirty minutes between doses.

The drug should be stopped when uterine contractions start. Too violent or long continued contractions must be controlled by the administration of an anesthetic. If the tetanic contractions cause fœtal distress and delivery is imminent, labor should be terminated by forceps.

The drug may also be administered as recommended by Hofbauer, e.i. by the use of nasal packs saturated with 0.5 cc. of the solution. The cotton may be removed and the nose flushed with saline solution if the contractions become too violent. This method of adminstration has been considered as dangerous by De Lee.

In use for third stage of labor ½ cc. of extract should be administered intramuscularly into the deltoid.

In cases of postpartem hæmorrhage 1 cc. should be given intramuscularly.

The routine of administration of dosage in medical inductions has been mentioned previously—and in addition to other substances ½ cc. of pituitary extract is given intramuscularly every half hour X 6 or till pain begins.

XII SUMMARY:

1. Proprietaries such as thymophysin, thyuitary, pituthymin and combinations with quinine are not to be recommended. They are diluted solutions of posterior pituitary or are the drug disguised.
2. Posterior Pituitary Sulfonate has been recommended as less dangerous than pituitary extract itself.
3. Pituitary extract augments the forces of labor which inflict such damage as lacerations of the cervix, vagina, perineum, even rupture of the uterus and cerebral damage in the babies.
4. Pituitary extract may cause severe shock and anaphylaxis.
5. There is a definite medico-legal aspect to the administration of a potent and dangerous drug as a matter of convenience.
6. Proper indications for pituitary extract are numerous in pregnancy to induce labor, in labor, and postpartem. These indications are well defined.
7. There are several definite contraindications to the use of pituitary extract.
8. Pituitary extract should not be used in normal labor, other than in the third stage of labor.
9. Dosage and method of administration must be considered for each different indication.
10. Much of the original enthusiasm which accompanied the introduction of pituitary extract has been carefully exploited by many and found to be dangerous.

XIII CONCLUSIONS:

The introduction of pituitary extract more than twenty-five years ago as a means of initiating and reinforcing labor pains constitutes a landmark in the development of the science and art of obstetrics. It has since been widely used during improper conditions and has caused much damage to mother and child. It must be used with scrupulous care and only for bona fide indications

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INFLUENZA

S. E. O'BRIEN 4T7

In view of the very high incidence of influenza in epidemic form, the author has attempted to prepare a brief survey of our present knowledge and understanding of this extremely infectious disease.

Definition:

Influenza is a highly contagious, acute, infectious disease occurring characteristically in epidemic and pandemic outbreaks. Its principal manifestations are a sudden onset, fever, marked prostration, aching pains in the back and limbs, and catarrhal inflammation of the upper respiratory tract.

Etiology:

Previous to the notorious 1918 outbreak, influenza was believed to have been caused by the Hæmophilus influenzæ (or bacillus of Pfeiffer). This organism is a small Gram-negative, non-motile bacillus found chiefly in the upper respiratory tract of man.

However it has since been proven beyond all doubt that the disease is caused by a filterable virus and that the Bacillus influenzæ is only a secondary invader. Moreover this bacillus has also been found as a secondary invader in many other infectious diseases such as whooping cough, diphtheria, chicken pox, and scarlet fever.

Experimental evidence that a filterable virus is the cause of influenza was first established in 1919. At that time, Nicolle and Lebailly discovered that the filtrates of nasal secretions from persons with this disease, when transmitted to other humans and also to certain species of monkeys by nasal and conjunctival innoculation, produced in these individuals the symptoms of influenza.

Soon after this, de la Rivière injected the filtered blood of influenza patients into himself and became definitely ill with influenza-like symptoms.

Other workers also reported similar findings—that the disease was transmitted by a filterable agent.

The virus was finally isolated in 1933 by Smith, Andrewes, and Laidlaw, who produced influenza in ferrets by the injection and nasal instillation of filtrates from throat washings of early human cases. It is thus evident that the influenza virus lodges in the upper respiratory tract and is most likely transmitted by droplet infection.

The relationship between the etiology of pandemic influenza and that of epidemic influenza is not thoroughly understood. It is thought that they are caused by different strains of the virus, but as yet complete knowledge regarding this is lacking. However, two different viruses have already been isolated—the influenza

A virus and the influenza B virus—and it is highly probable that there are more different strains.

Further work in this field has included the cultivation of the virus on chick embryo tissue and its isolation for study under the electron microscope. The isolated viruses obtained in this manner have been injected into humans who consequently contracted influenza.

It should be noted that although the specific causative organism in influenza is known, we know very little about the predisposing causes. The disease does however show a fairly marked seasonal incidence, with the majority of the epidemics occurring in the winter months.

One attack of influenza does not confer permanent immunity to subsequent infections. However it has been shown that following each attack, antibodies are produced in the blood, and that these provide a temporary immunity, both in experimental animals and in humans, for a period of several months.

Epidemiology:

The epidemiology of influenza and the history of the great world-wide pandemics is a study of great interest, but only a brief account can be given here.

The last great pandemic occurred after the first World War, from 1918 to 1920. The toll of lives during this pandemic was tremendous, with 20 million deaths reported out of a total of two hundred million cases. The deaths were due to a rapidly fatal influenzal pneunomia.

The characteristics of influenza pandemics have been summarized by Leichtenstern on the basis of the 1918-1920 pandemic:

(1) The disease appears in true pandemic waves, usually three waves, the second wave having the highest mortality.

(2) The waves travel with extreme rapidity all over the globe, from an origin in one part of the world.

(3) It is characterized by sudden mass infection.

(4) It is rapidly burnt out after several weeks in one locality.

(5) It is independent of season or weather.

(6) It begins at first with a high morbidity and a low mortality.

(7) Susceptibility is only slightly influenced by sex, age, and occupation. Usually the urban areas are stricken more severely than rural districts, however.

Epidemic influenza occurs much more frequently than the pandemic type, epidemics breaking out quite regularly every two years. It differs from pandemic influenza by being much less severe and having a lower morbidity rate—approximately 20% of the population. The severe, rapidly fatal influenzal pneumonia characteristic of pandemic outbreaks, is rarely seen in epidemic forms.

Pathology:

The pathological changes produced by influenza are very similar to those of any other acute catarrhal inflammation of the upper respiratory tract.

Rhinitis and pharyngitis are fairly constant, and there is usually an intense hæmorrhagic trachea-bronchitis. The characteristic changes in the trachea and bronchi are desquamation of the ciliated epithelium and infiltration of the mucosa with lymphocytes and plasma cells. According to Boyd, this epithelial desquamation leads the way to secondary invasion of the bronchi, as manifested by purulent exudate.

In severe cases there is extension of the inflammatory process upwards to involve the paranasal sinuses and downwards to the bronchioles and lung tissue. Involvement of the lungs takes the form of a bilateral bronchopneumonia, sometimes becoming confluent, and often characterized by profuse hæmorrhagic œdema. These cases are due to secondary infection of the lungs by Streptococcus hæmolyticus, Staphylococcus aureus, Hæmophilus influenzæ, or Pneumococcus.

Symptoms and Clinical Picture:

The incubation period of influenza is brief—usually about 48 hours—and the onset is sudden, with chills, marked prostration, and aching pains in the back and limbs. The symptoms reach their height in 24 hours. The temperature rises rapidly and ranges between 102°F. and 106°F., with the average being about 104°F. There may be severe headache; and a dry hacking cough, coryza, and sore throat are fairly constant symptoms. In some outbreaks, gastro-intestinal symptoms may predominate. These include nausea, vomiting, diarrhoea, and abdominal pain.

The course of the disease is usually brief, averaging about three or four days. At the end of this time the temperature falls fairly rapidly and the symptoms disappear. Convalescence is usually prolonged and accompanied by depression and asthenia.

Physical examination usually yields little in the way of positive findings. The pulse rate and respiratory rate are slightly elevated and the face may be somewhat flushed and cyanotic in more severe cases. Inspection of the throat usually reveals some congestion of the fauces and pharyngeal mucosa.

Examination of the chest is usually negative, though a few scattered rhonchi and high pitched rales may be heard in those cases where pulmonary complications are imminent.

The white blood count is low, illustrating the leucopenia characteristic of most virus diseases. Culture of the blood is usually sterile.

Persistence of the fever and the appearance of chest signs after the first 3 or 4 days indicate complications. An increase of the white blood cells above normal is indicative of secondary invasion by pyogenic organisms.

Differential Diagnosis:

The diagnosis of influenza, except in times of epidemic, is a precarious one, as French points out. It should only be made after physical examination and laboratory investigation have ruled out more typical and more serious diseases. In many cases the diagnosis may resolve itself into the problem of differentiating

between the many acute infections accompanied by marked fever, and only the more common of these can be indicated here;

(1) Common cold—This is usually more localized and accompanied by much less severe degree of prostration, but in severe colds the differentiation may be difficult.

(2) Lobar Pneumonia—Usually accompanied by more or less typical chest signs and pleuritic pain.

(3) The acute exanthematous diseases (simple measles, German measles, scarlet fever, chicken pox, and small pox). The appearance of the characteristic rash indicates the true diagnosis.

(4) Infectious hepatitis—often has an influenza-like onset, but the appearance of jaundice usually makes the diagnosis evident.

(5) Poliomyelitis in the early stages: season, incidence of other cases in the district, and the onset of paralysis differentiates this from influenza.

(6) Acute Rheumatic Fever—The flitting polyarthritis, cardiac signs, and the more prolonged course are characteristic.

(7) Primary atypical (virus) pneumonia—this disease runs a more prolonged course and the symptoms are less severe than in influenza.

(8) Meningitis—Onset of neck rigidity, the elevated white blood count, and the spinal fiuid findings differentiate early meningitis from influenza.

(9) Subacute bacterial endocarditis—The clinical picture of this condition may be quite similar to influenza during its early period, but the history of previous cardiac damage, the prolonged course, embolic manifestations and positive blood culture make the diagnosis evident.

These are only some of the many conditions which may simulate influenza, especially in their early stages, but they are the common ones which should always be considered by the practitioner when he is confronted with such a case.

Treatment:

There is no specific cure for influenza in general use as yet. The sulphonamides and penicillin have been found to be ineffective in so far as the primary disease is concerned. However, results reported on complicated cases, are sufficient to warrant their use wherever severe secondary infection, such as bronchopneumonia, makes its appearance. Some workers have reported dramatic results in a few cases treated with human convalescent serum. The serum was used mainly in severe, moribund cases and much further work is indicated before its value can be properly assessed.

At the present time therefore, we must be content with purely symptomatic treatment in the uncomplicated case of influenza. Complete rest in bed is essential for a period extending well into convalescence. The diet should be light during the febrile stage and large quantities of fluid should be administered daily. Other

palliative measures include the use of an ice-cap or aspirin (10 grs.) for headache, sponge baths for high fever, barbitone for sleeplessness, and the inhalation of steam or administration of Dover's powder (5-10 grs.) for the relief of cough.

Prophylaxis:

The prevention of influenza has, up to now, been a perplexing problem. The general measures adopted in all infectious diseases should be employed for influenza also. These include the isolation of the patient from contact with the other members of the household, the proper handling of contaminated articles, and the avoidance of crowded rooms and public places during the time of epidemics.

Much attention has been centered lately on influenza vaccines. These vaccines are prepared by inoculating chick embryos with the influenza virus, and, after an incubation period of 48 hours, drawing off the infected allantoic fluid. This fluid is then used for immunization purposes.

The vaccine is still in its infancy however, and has not yet been adopted for general use. However encouraging results were published by the United States Army in 1944. The effect of the vaccine was studied on a group of approximately 12,500 army recruits at the time of an influenza epidemic. Subcutaneous injection of a vaccine was given to 6,263 men, while 6,211 others received injections of sterile water as a control. In the vaccinated group there were 138 cases during the epidemic which shortly followed, while in the control group there were 422 cases.

Though these results seem hopeful, there are many problems yet to be overcome before vaccination against influenza comes into general practice. Such problems as the duration of immunity following vaccination, and the efficiency of one vaccine in the face of several strains of influenza virus, have yet to be solved.

Prognosis:

The prognosis of a simple uncomplicated case of influenza is excellent, complete recovery being the rule. In pandemics, believed to be caused by a more virulent strain of the virus, the prognosis is less favourable, death occurring in many instances from severe, secondary influenzal pneumonia.

The prognosis depends mainly on the general condition of the patient. Those persons who insist on being up and around during a bout of influenza are more prone to develop secondary infections, than those who take the sensible precautions.

Complications:

Influenza in itself is not serious disease, but it so lowers the general and local resistance of the body that secondary infection may readily occur, often with a high rate of mortality.

The most severe of the secondary infections is the bronchopneumonia mentioned above. It should be suspected when the fever and chest signs persist or increase in severity after the first 3 or 4 days, and also if the white blood count

becomes elevated. In such instances, chemotherapy should be administered promptly.

The pneumonia itself may be complicated by empyema, lung abscess, and bronchiectasis, while pleurisy is relatively uncommon.

The other common complications include purulent bronchitis, laryngitis, sinusitis, and otitis media which occurs chiefly in children. Meningitis may also be seen complicating influenza.

The author wishes to express his gratitude to Dr. R. B. Kerr for his helpful suggestions and valuable criticisms of this article.

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LIVER FUNCTION TESTS—PART I

By R. D. HAWKINS

It has become increasingly apparent that the efficiency of the liver may be seriously impaired not only in diseases affecting this organ, but in other conditions as well. To determine the presence and degree of hepatic insufficiency which may escape detection in the ordinary clinical and laboratory examination of the patient, numerous liver function tests have been proposed. These tests indicate the existing functional state of the hepatic cells but do not necessarily reveal the cause of the impaired function, but if one knows from clinical laboratory or X-ray studies the general nature of the pathological condition, then the results of liver function tests are of unusual value.

Many of the liver tests are too complicated for general use; others have proved to be insensitive or inaccurate or both; still others may actually be harmful in that they require the administration of drugs which may impose an additional strain on an already damaged liver. Thus, many have failed to measure up to the exacting practical criteria and have gradually fallen into disuse; some have survived and have been found to be clinically useful, although here too the claims of individual enthusiasts were generally found to be exaggerated; in the course of time, however, their field of usefulness and their limitations have been fully realized and they now occupy a definite place in the laboratory investigation of hepatic disease.

The purpose of any particular liver function test is of the utmost importance. It may be designed (1) to assist in the differential diagnosis of intra-hepatic and extra-hepatic disease; (2) to ascertain the activity of the disease and its response to therapy or to follow the progress of a chronic hepatic disease; (3) to aid in the detection of hepatic insufficiency. Liver function tests in addition may be used in patients for whom major surgery is planned, as a means of guiding pre-operative therapy and thus they may help to estimate the risk involved in such surgery; they may also aid in the detection of subclinical liver damage as in people exposed to hepato-toxic agents such as chloroform or in patients treated with hepato-toxic drugs such as arsenicals.

When one considers the multiplicity of functions of the liver, one must realize that no single test can possibly be expected to reveal everything about the function of that organ and no single test should therefore be accepted or condemned from such an unreasonable standpoint. To appraise in a satisfactory manner the functional status of the liver it is therefore desirable to test as many functions as possible. A good deal of the present dissatisfaction with the results obtained with liver function tests is due to a tendency to rely solely on one test. No correlation has been shown between the type of pathological lesion present and the function which is disturbed, nor is there any definite index clinically as to which function is disturbed first.

Three physiological factors limit the quantitative estimation of hepatic insufficiency:

(1) The duration of the disease; in many conditions the pathology concerned is complicated; although jaundice may be predominantly obstructive, hepatogenous or hemolytic, pure forms of each seldom occur after jaundice has been present for a period of time. The duration of the disease is therefore a primary consideration in the use of liver function tests.

(2) The relatively large hepatic reserve: it is impossible to test the vital function of the liver, though many of its activities can be tested individually. How much of a factor of safety must be functionally or anatomically suppressed before a liver function test is useful is uncertain. Some of the earlier tests have been evaluated upon the basis of the results following the removal of increasing amounts of liver, but many have not been tested by this crucial method. The relatively large reserve of hepatic tissue is fortunate for the patient, but unfortunate from the view-point of the physiologist who wishes to design a quantitative test for the extent of hepatic reserve.

(3) The regenerative capacity of the liver: unless the liver damage is extensive and rapid, simultaneously occurring regeneration is likely to compensate for, and mask the effect of the destructive changes. These three factors demonstrate the need of testing more than a single activity of the liver.

It is important to the worker performing the tests to know the limitations of each liver function test—for example, there is no point in testing with dyes in the presence of a hyperbilirubinemia with a direct van den Bergh, nor or doing a bilirubin clearance test in the presence of a hyperbilirubinemia. It is only through an understanding of the physiological principles upon which each test is based that errors in interpretation can be avoided and an understanding of the limitations be appreciated.

It is impossible within this short period of time to evaluate critically the innumerable tests for liver function that have appeared within the past three decades. Therefore, it seems best to confine this discussion to an explanation of the principles, the advantages and disadvantages of a group of tests which have been chosen arbitrarily, but which now occupy a definite place in the laboratory investigation of hepatic disease.

Liver function tests can be grouped under the following headings:

A Tests based on a study of the function of the liver with respect to the excretion of the biliary elements:

* (a) Van den Bergh test

(b) Icterus index

* (c) Urobilinogen determinations in urine and fæces

* (d) Analysis of the products of duodeno-biliary drainage

(e) Estimation of the extent of bile acid production and of the elimination of bile salts

B Tests based on the Detoxicating Mechanism of the Liver.

* (a) Oral and intravenous tests for synthesis of Hippuric acid
(b) Cinchophen oxidation test.

C Tests based on the role of the liver in intermediary metabolism.

* (a) Protein
* (b) Carbohydrate
* (c) Fat and cholesterol

D Tests based on the excretory function of the liver.

(a) Rose bengal clearance test
* (b) Bromsulphthalein clearance test
(c) Bilirubin clearance test

E Miscellaneous liver function tests.

* (a) Measurement of Coagulation Factors
(b) Colloidal Gold reaction
* (c) Serum phosphatase determinations
(d) Takata-Ara test
* (e) Cephalin-Cholesterol flocculation test
(f) Thymol turbidity test

*Tests discussed in this paper.

A. The Tests Based on the Excretion of the Biliary Elements

VAN DEN BERGH TEST

The earlier work of Mann, Bollman and Magath on hepatectomized animals indicated that the liver acts as an excretory organ with respect to bilirubin just as the kidney does for urea. Hemoglobin, through the action of the reticulo-endothelial cells of the liver, bone marrow, spleen and general connective tissue undergoes a series of chemical transformations in the course of which iron is released and the pigment bilirubin type I (hemobilirubin) is produced and taken up by the blood stream. The circulating bilirubin is then removed by the parenchymal cells of the liver and excreted into the bile as bilirubin type II (cholobilirubin). The intestinal cholobilirubin is transformed by bacterial action into urobilinogen, most of which is excreted with the fæces. Part of the urobilinogen is absorbed from the intestine into the blood stream and on reaching the liver is removed by the parenchymal cells and excreted into the bile as bilirubin type II. Traces of urobilinogen are present in the blood and appear normally in the urine.

The presence of icterus indicates an increased concentration of bilirubin in the blood, a condition which can be produced by three general types of disturbance, acting singly or in combination;

(a) *hemolytic* jaundice, that is, cases in which bilirubin is produced in excess of the capacity of the liver to excrete it (jaundice of the newborn, malaria, black water fever and pernicious anemia are examples of such a condition);

(b) *hepatogenous* jaundice, the condition in which the rate of production of bilirubin is not increased but because of toxic or infectious injury to the liver cells and finer bile passages, bilirubin accumulates in the blood stream through an

impairment of the excretory function of the hepatic cells—cirrhosis, carcinoma or abcess of the liver and acute and toxic hepatitis result in this form of jaundice;

(c) *obstructive* jaundice, the condition where obstruction to larger bile passages causes a reflux of bilirubin into the blood; under such conditions the bile excreted in the usual way cannot escape into the intestine and is reabsorbed into the blood stream; this form of jaundice may occur as a result of blockage of the common bile duct by a stone, a stricture or by inflammation, or as a result of carcinoma of the common duct or of the head of the pancreas.

Van den Bergh and Müller on the basis of Ehrlich's diazo reaction were able to distinguish two types of reaction with bilirubin; in cases where bilirubin had passed through the liver, an immediate so-called direct reaction was obtained, whereas in cases of hemolytic jaundice, where the bilirubin had *not* been excreted by the liver cells, a reaction was obtained only after treatment with alcohol—the so-called indirect reaction with the qualitative Van den Bergh. Bilirubin type II thus gives the direct reaction, wher type I reacts less rapidly so that a violet colour does not appear for some time or not until after preliminary treatment with alcohol. A third reaction, the so-called delayed or biphasic response may be found in certain types of jaundice believed to be due to the presence of both types of bilirubin.

According to the usual view, most of the normal bilirubin in the blood is hemobilirubin, which cannot penetrate the renal filter—bilirubin type II can pass through the renal filter and appears in the urine when its concentration in the serum is about 2 mg./100 cc.; however, there is doubt now whether bilirubin type II behaves as a threshold substance and it has been suggested that the kidney does excrete it regardless of its concentration in the blood. Nevertheless, with the ordinary qualitative tests, it is only found when the blood level is greater than normal.

High values for serum bilirubin are found in acute severe hepatogenous forms of jaundice and in neoplastic biliary obstruction; intermediate values in milder degrees of hepatic parenchymal lesions and in conditions in which intermittent or partial biliary obstruction exists; low grades in the presence of direct Van den Bergh are found in portal cirrhosis and syphilitic hepatitis, in the presence of a stone in the duct and in infectious forms of cholesystitis. The serum bilirubin may be measured quantitatively by the photoelectric method of Malloy and Evelyn.

The quantitative Van den Bergh is not reliable for differential diagnosis of extra-hepatic obstructive jaundice from the parenchymal type. Most workers seem to agree that the quantitative test is of value in confirming the diagnosis of hemolytic jaundice, where the positive indirect test is obtained.

The quantitative test is of value as an index of an increase or decrease in the intensity of jaundice which cannot be followed by clinical observation. It is also of value in latent or subclinical jaundice, especially that associated with sulfonamide therapy. It is definitely superior to the icterus index as a measure of bilirubinemia, since hemolysis and carotenemia do not interfere, but a single quantita-

tive determination has limited diagnostic value and cannot be considered a sensitive test of liver function, since marked chronic liver disease may exist with normal bilirubin levels.

Urobilinogen Determination in Urine and Faeces

Elman and McMaster have demonstrated that bile pigment on reaching the intestine is converted by bacteria to urobilinogen, the major part of which is excreted, while the remainder is absorbed through the intestine and brought to the liver by the portal circulation. The liver will convert most of this urobilinogen to bilirubin, while the rest is excreted in the urine as urobilinogen which may be oxidized to urobilin.

Condition	Bilirubin in *urine*	Urobilinogen (24 hours) in urine	Urobilinogen (24 hours) in faeces
Normal	—	+ (0-4 mg.)	+ (40-280 mg.)
Uncomplicated *hemolytic* jaundice	—	++ (5-30 mg.)	++++ (600-2000 mg.)
Uncomplicated *hepatogenous* jaundice	+	++ (0.5-100 mg.)	+ (10-30 mg.)
Uncomplicated *complete extra-hepatic obstructive* jaundice	++++	— (less than 0.5 mg.)	— (less than 5 mg.)

Changes in Pigment Metabolism which occur in *uncomplicated* forms of jaundice

The preceding table shows the variations which can occur in the urobilinogen levels in various forms of uncomplicatel jaundice. In hemolytic jaundice bilirubin type I increases beyond the capacity of the liver to excrete it and the amount of this fraction in the blood rises as a result. The liver compensates in part by excreting greater amounts of cholobilirubin into the bile and more urobilinogen is formed in the intestine; as a consequence, more is absorbed into the blood stream, with the result that urobilinogen appears in the urine in increased concentration. Since the increase in the serum is in bilirubin type I, the urine will contain no bilirubin. Therefore in hemolytic jaundice there is an increased concentration of urobilinogen in the urine and fæces and there is an absence of bilirubin from the urine.

In diffuse hepatitis, there will be decreased pigment excretion in the bile and of the amount which does reach the intestine and is converted to urobilinogen at that point, little of the resulting urobilinogen absorbed can be excreted by the damaged liver cells, so that more urobilinogen than normal appears in the urine. There will be increased concentration of bilirubin type II in the blood as a result of rupture following obstruction of the finer capillaries, but most of this does not reach the intestine in the early stages of the process. Because of the increase in

serum bilirubin type II above the threshold level bilirubin appears in the urine. In more severe types of hepatic jaundice, obstruction of the intra-hepatic ducts is more marked, so that less bilirubin type II reaches the intestine with a consequent reduction in urobilinogen production. Therefore in the later stages of hepatic jaundice, urobilinogen may completely disappear from the urine, but as recovery proceeds, bilirubin type II is once more excreted into the intestine but the urobilinogen which is reabsorbed cannot be excreted at the normal rate by the cells which are still damaged; as a result, the urobilinogen concentration in the urine rises above normal. In hepatic jaundice, therefore, there is an increased amount of bilirubin and urobilinogen in the urine in the early and in the recovery stages, but only increased amounts of bilirubin in the fully advanced stage.

In complete obstructive jaundice, bilirubin type II cannot be excreted into the intestine and no urobilinogen is formed. The duodenal contents therefore do not contain bile pigments. Eventually the bile capillaries and the ducts above the obstruction dilate and rupture, permitting regurgitation of this type of bilirubin into the blood and lymph with a consequent increase in the concentration of bilirubin in the serum. Because this type of bilirubin can pass through the renal filter, it appears in the urine. Thus extra-hepatic obstructive jaundice is characterized by the presence of bilirubin in the urine and the absence of urobilinogen from the urine and fæces.

The difficulty in differentiation is that obstruction may not be complete and secondary hepatitis may be present, and in hepatic jaundice there may be marked obstruction of the intra-hepatic ducts. In incomplete obstructive jaundice some bilirubin type II will reach the intestine and the reabsorbed urobilinogen will appear in the urine in increased concentration; therefore, the urinary findings are not unlike those in hepatic jaundice. Moreover, in about 10% of the cases of hepatic jaundice, the severity may result in an almose complete intra-hepatic obstruction; in such cases no bilirubin will be found in the intestine, no urobilinogen in the urine and the findings will be the same as those in extra-hepatic obstructive jaundice.

Watson has an elaborate quantitative method for the determination of urobilinogen in the urine and fæces, but Sparkman's method is more rapid and simple and yields values which check with those of Watson for all practical purposes.

While many workers consider the urobilinogen test to be more valuable than any of the other tests they have studied for the diagnosis of complete external obstruction, certain limitations are known to reside in the test:

(1) Tests on single urine and fæcal specimens when assayed by quantitative or qualitative methods, even though helpful, are of equivocal value, and from a practical standpoint, because of associated illness and limited diet, stools, particularly four day specimens as recommended by Watson, are frequently not obtainable.

(2) In the differential diagnosis of jaundice, the urobilinogen results may be modified and their diagnostic value annulled by complications such as infection of

the liver and bile passages, and in anæmia. In the former case, the organisms along infected passages may convert the bile pigment to urobilinogen which will be carried by the blood stream to the kidneys and thence excreted.

(3) Urobilinogen may sometimes be formed in other tissues than the intestine and infected liver; it may be excreted excessively in splenic and pulmonary infarction and in hematomas in various locations.

Duodenary-Biliary Drainage

The examination of the products of duodenal drainage for enzymes, bile pigment, bile salts, pus cells, crystals, epithelium, bacteria and parasites can yield much diagnostic informatioon of significance. However, to obtain this information considerable careful chemical and microscopic work is required and the procedure and work has to be conducted by an experienced person, one who knows more physiology and pathology than the ordinary technician. This fact, unfortunately, limits the practicality of this test.

The presence or absence of bile pigment is of obvious diagnostic importance as is an estimation of its amount. The presence or absence of bile salts and the amount is also important. Cholesterol and calcium bilirubinate crystals provide information regarding the existence of stones in the duct or gall bladder; blood, in the absence of a duodenal ulcer, may assist in the recognition of an ulcerating carcinoma involving the ampulla. The presence of an abundance of epithelial debris, pus cells and bacteria may indicate catarrhal inflammation of the bile passages; lightly pigmented bile and no crystals are found in the duodenal contents in acute hepatitis. A simultaneous study of the enzymes may reveal important information regarding the pancreas.

Of the serious drawbacks which prevent this means of evaluating liver from enjoying a wider use the following should be mentioned:

(1) Considerable skill and care are required in the technique of duodenal intubation.

(2) Often the patient is too ill to be bothered with the manipulation necessary in such a procedure.

(3) Pernicious anæmia, gastric anacidity and disease of the pancreas must be considered in the interpretation of the results from duodeno-biliary drainage.

B. Tests Based on the Detoxicating Mechanism of the Liver

THE ORAL AND INTRAVENOUS TEST FOR THE SYNTHESIS OF HIPPURIC ACID

The ability of the liver to remove from the circulation various noxious substances, alter them chemically, or combine them with certain common constituents of blood in such a manner as to render them physiologically inert has been known for years. Various clinical tests with such substances as thymol, menthol, camphor, salicylates and phenol have been used but they have not been entrely suc-

cessful. The only survivor of such tests of the detoxicating function is one that is based on the ability of the liver to conjugate benzoic acid and amino-acetic acid to form hippuric acid.

Quick has pointed out that the synthesis of hippuric acid in human beings seems to depend largely on the rate at which the liver can supply amino-acetic acid; he has demonstrated that the maximum amount of hippuric acid which can be synthesized is limited, practically constant, and independent of the exact amount of sodium benzoate injected or ingested, provided that the amount of benzoic acid is in excess of the capacity of the liver to conjugate it all, with the glycine within a fixed period of time. The product of conjugation, hippuric acid, is eliminated in the urine and the estimation of the amount eliminated may be done by precipitation and weighing or by an ether extraction-formal titration method.

In certain types of liver damage the output of hippuric acid, which will be correspondingly diminished because of the lack of glycine to combine with the benzoic acid, will serve as an index of liver damage. In the test in which 5.9 grams of benzoic acid is administered orally, the excretion of 2.6-3.3 grams of hippuric acid is the standard for a normal individual. Quick reported a decreased output occurred in cases of catarrhal jaundice, various forms of acute hepatitis and in hepatic cirrhosis, but the autput usualy was normal in cases of cholecystitis and cholelithiasis.

Snell and Plunkett emphasize that low hippuric acid values cannot be used as a criterion of imparied liver function if there is sufficiently marked renal impairment to cause elevation of blood urea, if the patient is dehydrated to a point where water elimination and urinary volume are reduced, or if gastric retention exists. Moreover, obstruction in the urinary tract—enlarged prostate, for example—may retard elimination of synthesized hippuric acid and result thus in low hippuric acid output. To exclude a possible factor of delayed elimination of the kidney, Kohlstaedt and Helmer suggested that a simultaneous study of urea clearance should be made to differentiate reduced synthesis with normal renal excretion and normal synthesis with reduced renal excretion.

To overcome some of the difficulties attending the oral hippuric acid test Quick and his associates in 1938 devised an intravenous modification of it. This modification involved the intravenous administration of 1.77 grams of sodium benzoate and the collection of urine for one hour after the injection. Lipschutz in 1939 also outlined an intravenous modification which Quick has stated is open to two criticisms. Lipschutz's method measures the percentage of administered benzoic acid which is excreted as hippuric acid, whereas Quick's intravenous method measures the rate at which a particular liver can synthesize hippuric acid. Quick maintains that there is no evidence that the percent eliminated has anything to do with liver function impairment—it is the rate of formation which is important. Moreover, the dose of benzoic acid has no effect, the excretion depending on the liver glycine stores, and benzoic acid must be given in excess of the

liver's capacity to conjugate it within a fixed period. It is the one-hour sample which reflects the maximum capacity of the liver and therefore Quick's one-hour intravenous method is a more sensitive test in detecting impairment of liver function than the test devised by Lipschutz, in which two hours is the length of time in which the liver is allowed to act.

The hippuric acid test in general has the following advantages:

1. It is a simple, sensitive and reliable index of hepatic insufficiency;
2. It is applicable in jaundice and non-jaundiced patients;
3. The intravenous method measures maximum hepatic worp for a short period, whereas the oral method allows four hours for the slightly impaired liver to overtake the normal liver in the amount of work performed;
4. The intravenous route of administration eliminates all question of absorption, insures accurate dosage and requires a small dose of the benzoate; it also avoids difficulties with vomiting and diarrhœa;
5. Boyce and McFetridge found that the oral test is useful in determining the proper course of pre-operative and post-operative procedure. There is, however, considerable difference of opinion as to the value of the hippuric acid test as a diagnostic aid in determining the type of jaundice.

The subject of hyperexcretion of hippuric acid is at present a matter for dispute. Rosenberg and Soskin contend that excessive execretion signifies early hepatic damage on the basis of "hyperexcitability of the damaged liver cells". Hepatoxic agents have been shown experimentally, to produce first an increase in the irritability of that organ, thereby augmenting the rate at which the organ performs its various functions. Rosenberg suggests that the hyperexcretion of hippuric acid may be another manifestation of such a hyperirritable phase.

Part II of this paper will be included in the March issue of volume XXIII

Feature

L. ROGERS, Editor

HOW TO WRITE EXAMINATIONS — AND PASS

By J. HAROLD COUCH, M.A., F.R.C.S.,
University of Toronto Department of Surgery

Examinations are a poor way of determining what a student knows. No one can expect to fathom the depths, often abysmal, of a medical student's mind, by setting a paper every year containing five questions and reading the reams of drivel he submits in the vacuous hope of hiding his ignorance.

Examinations are a nuisance. Maybe so, but that is exactly the reason why they are worthwhile. Life is made up of one crisis after another and for many they do not even wait their turn. As one matures, the problems become increasingly serious, ranging from the lost jacknife of childhood to the loss of loved ones, business, ambitions, even lost illusions, as the years roll on. Examinations, instead of being looked upon as obstacles to youthful bliss, should be considered as practice runs in preparation for life's bigger races, which can be creditably run only by those conditioned to the test. The abolition of examinations in public schools and the substitution of the "recommend" system was one of the most cruel and short-sighted steps in education in the past 100 years. I hope my youngsters never are recommended and have to write many examinations every year for then they may be able to take the tougher things of life less seriously.

Examinations are not primarily to find out how much you have learned. They are a form of education. It is *how* you stand up to them that matters; that tells us what sort of material you are; and what sort of citizen you are likely to become. Are you the type of person who should be stamped as a professional man in the community? Will you make a good, all round, helpful doctor? Never mind how much you know or what you know. Most of it will be out of date by the time you start practice, anyway. In fact, your knowledge and views will, in many instances, be quite wrong and unacceptable right now, even before you graduate, if you happen to run foul of an examiner from some medical school other than the one in which you have received your teaching.

And yet, you want to make a decent showing in this particular test just as members of the track team or rugby teams want to make good for dear old Alma Mater—not to mention poor old Pater and Mater back home, who perhaps had to pinch a little to pay your University fees and also give you enough to sport a taxi for the Med-at-Home. Besides, in case you are interested, if you fail too many examinations you are likely to be thrown out.

So, if you are going to try examinations, why not enjoy them? Get as much out of them as you can, and *pass!* Nothing could be easier. Goodness knows the poor old examiner doesn't want to plough you. He hopes to goodness he can push you through in spite of your frantic efforts to keep him from doing so.

Every time I have to mark a question on a paper or, worse still, set and mark the whole business, I am conscious of a rising indignation at how hard you make it for the examiner to pass you. Why do you do it?

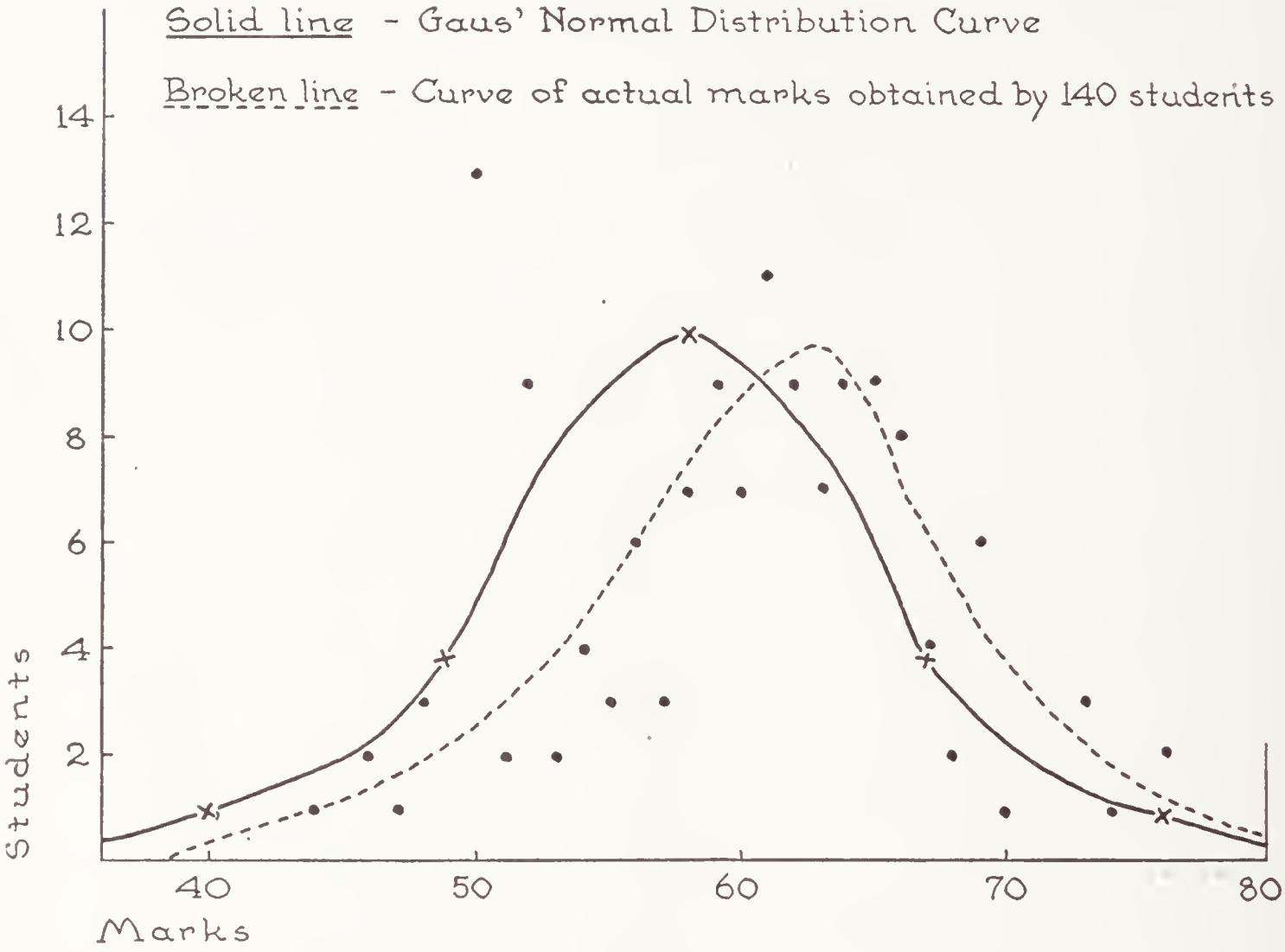

FIG. 1

Know Your Stuff

It is, of course, helpful, in writing examinations successfully, to know what you're writing about. But even so, many a lad who knows his stuff has failed; many more who did not know so much have passed. However, the student who can attend lectures and clinics and grasp what the lecturer is trying, often unsuccessfully, to get across, is more likely to have a fairly good type of mind which will make itself apparent in the manner in which he presents his material.

Answer the question that is asked

To answer the question as it is asked you must *read* the question word by word. Suppose the questions reads "Describe the gross apearances at the site of a fracture during various stages of healing". Most students glance at it, see the words "fracture" and "healing" and spill pages about osteoblasts and osteoclasts which are not gross but can be seen only with a microscope. The question asks for *gross* appearance NOT "process of healing". Do not re-word the question; it annoys the examiner. Don't say, "Oh, the poor old coot means such and such but didn't word it very clearly". Your evaluation of his mentality may be correct but this is hardly the time to rub it in.

If the question reads "discuss" so and so, that means spill all you want to, we are not going to read it in detail, anyway, but are going to mark you on the other questions and have put this one in just to keep you here till the bell rings. If the question asks you to "list" something such as signs and symptoms, then list them, don't discuss them. If the question asks you to "tell all you know," just write "nothing" and go on to the next question. You are entitled to full marks on that question since the examiner can't call you a liar—besides your answer is probably correct, anyway.

If you do not answer the question as asked the examiner is entitled to draw a big red line through it and give you no marks at all. And he may be mad enough when he comes to your paper to do it.

If, of course, you are caught out and know nothing of the question, you may have to bluff a little and try to get the old boy to let you re-word it. Such a state of affairs reminds us of the Theolog. from Emmanuel College who got a tip that a question was to be on the paper concerning the "Kings of Israel". To his chagrin the question was instead "List the Prophets of Israel". In his answer he wrote "Far be it from me to discuss these Holy Prophets, but the Kings of Israel are these." Pretty poor! It won't work.

Margin

Leave a neat margin. Where do you think the examiner is going to write down the marks he gives you? If you want marks, leave him some place to put them. If you don't expect or want any marks, then, of course, you need not bother with a margin. Surprising how many leave no margin.

Follow The Order

Answer the questions in the order they are asked. This makes a good impression. Looks as though they are all equally easy for you! No choice! Doesn't matter what comes first! Also makes it easier for the examiner and that pleases him. We hear a lot of optimistic chat about picking out the one you know best and writing a long answer to impress the examiner with how good you are. It doesn't work that way. He figures that if you know one better than another, then you must know the other worse than the one. It tells him at once that you are not too sure—one of those birds who is just on the line—may get through if we happen to ask the right questions. If you go ahead quietly and evenly, taking them in your stride and in correct sequence you may slip a poor answer in with your better answers and get away with it. As soon as you jump a question, you proclaim in a loud voice that you are unhappy about it and we approach that answer with a firmly fixed, preconceived idea that it will be poor and worth very few marks.

Organize Your Answer

Perhaps this is the most valuable single tip. Read the question carefully, plan your answer clearly, in an organized fashion. Make headings 1, 2, 3, with subheadings a, b, c, d, e, etc. It isn't what you say but how you say it that is important.

There may still be a few examiners who think they are trying to find out what you know, but not many, because there are not many of your teachers on the Medical Faculty who have that stodgy old, school-teacher type of mind. Most of them recognize clearly that the prime function of a university is to cultivate the mind—not to teach a trade.

Of course, one has to be constantly on guard or a Medical Faculty (as well as Dentistry, S.P.S., Forestry, etc.) tends to deteriorate into a Trades School. The part of the teaching which may more correctly be called a "trade", probably requires a periodic "trade test" to ascertain what facts and skills the prospective tradesman may have acquired. But this is the only time examinations should be conducted to determine what the candidate knows.

More important, however, than the training of a tradesman is the making of a doctor. The problem is one of developing an entire personality. The ability to reason, to analyze, to weigh, to accept, to reject, to exercise judgment, is what makes a learned man rather than a gramophone record.

Any good gardener, if asked to produce a beautiful and bountiful garden, would choose a rich, well-cultivated piece of soil, even though it contained no seeds, rather than an uncultivated field of poor material, though it might contain thousands of dollars worth of seed thrown here and there without any plan or organization.

So in life, the cultivated and disciplined mind, free from a mass of fixed prejudices, will produce the most beautiful thoughts and the most useful ideas. Mere dogma is not enough to turn out a good doctor. Unless critical ability, judgment, analytical process, be developed, all the seed which we may sow will have fallen on barren ground, and will bring forth nothing.

Some seem to think education means putting stuff into your heads, whereas the exact opposite is the truth. The word education by its derivation, means a drawing out, or development, of what is already within, "e—duco—I lead out." (That's Latin.)

A third-class mind may remember and hand back something that has been given to it; a second-class mind may organize it; only a first-class mind can analyze it. Nobody hopes for that; but you see, if you do not at least organize your answer you put yourself below second class, or 60%, no matter how much you know.

However, 60% will get you through. In fact, we let you by if you earn half the total number of marks; that is to say, if you remember only 50% of the material taught during the year; and that in a muddled, hodge-podge kind of way. Pretty soft, eh?

If you had a keg and poured a hundred quarts of beer into it, and then turned the tap at the bottom, what would you expect to come out? Beer, of course. That is all we expect. But would you be satisfied to get back only 50 quarts of stale, flat beer? No! You would say that it was a bad keg, it leaked, and you would throw it away. Yet we let you through if you hand back only 50% of what was offered

to you even though it be flat, befuddled and poorly understood. What we should do is throw you out at once.

If, however, you got back from the keg better beer; beer that had worked; that had some kick to it, what would you think?—Well, that is the way we feel, too, whenever we encounter a paper that suggests the writer has thought over and digested and improved all that we have tried to tell him. Do you wonder then, that we are glad to give such a student first class marks?

Keep It Short

If you organize your answers you will have no need to fill two or three books with ramblings that are not going to be read, anyway. Usually the answers that get the highest mark in any group are contained in one book and often that book has one or two pages left over. A long answer starts off by making a bad impression on the examiner, though if he sees evidence of organization in it, he will not hold it against you.

And furthermore, please write legibly. True, this is not an examination in penmanship, but a well organized, neat type of mind is the kind you would want looking after you if you were ill. Certainly I should hate to have some of the sloppy, rough, pen-wielders stirring up my insides with a scalpel.

Plan Your Time

If there are 5 questions and you have 2½ hours or 150 minutes, that means 30 minutes are allotted for each question. Some may have two or more parts such as "Anatomy and Pathology," so allow time for *each* part. You can even count them as separate questions, and do not hesitate to separate them in your answers. Do not jumble them together. But remember that the chances are each question is worth about the same number of marks. Spend your time wisely. If you plan your time before you start, no paper is too long. There is no excuse for putting too much time on some questions and then being hurried at the last. Your scribbled "time-up" on the last page gets you no sympathy; only condemnation as a nincompoop without enough brains to organize your plan of campaign, either on an examination or anywhere else. So don't try to fool the examiners by writing "no more time" after scratching a few lines at the end of your paper on the question you know little or nothing about.

AEGROTAT

Don't ask for aegrotat standing unless your claim is genuine. Many times when a candidate applies for aegrotat, it is found that he has already failed in other subjects. If your claim is sincere, however, you can be sure of sympathetic consideration after a searching investigation.

Do The Best You Can

After every examination we hear loud wails from some that the questions were too hard. Nonsense. Some students got over 70% on the very paper you

are yelping about. The majority did reasonably well and a few failed. Just as it should be. It is not how tough the questions are but how tough the marking is, that determines the showing of the group. If all fail, the marking was too severe. If all pass it was too easy. A tough paper is your chance to show how you would approach and handle a tough job in life.

Normal Distribution Curve

As a matter of fact, marks of a large group, on an examination, distribute themselves according to the Gaus Distribution Curve, as does every other quality in nature. If the marks follow this curve, then it was a good examination, well marked, covering an average group of people. According to the curve, 5% should get first class honours, 20% should get second class honours, 50% (the great unwashed) stand about 3rd class, 20% barely squeeze through, and 5% fail.

Look at figure 1. The solid line shows the normal curve and the broken line the actual marks of 140 candidates on the last paper I set. Note that 5% or 7 students obtained first class rating, 5% failed, the others strung out just about as they should according to the normal distribution curve. And yet there were the usual complaints that the paper was too hard.

Now look at it again in more detail. Notice that the broken line lies a little to the right of the solid line. That is to say, the whole curve of actual marks leans a little more toward the upper marks than does the theoretical curve. This means that the marking was a shade easier than it should have been if the examiner had been a cold blooded scientist.

Look again at the dot opposite the 50 mark level. It is out of line—too many students got 50 marks. This means that we pushed a few up to 50 (just over the pass line) who should have received only 48 or 46 marks. And yet the students think we are hard-hearted. Remember also that all the papers were marked before the curve was plotted; and the marks are on record in the medical office right now, and can be checked at any time.

Conclusion

In conclusion, let me urge that you approach examinations with a common sense determination to comprehend the questions and to answer directly and thoughtfully. Furthermore, let me suggest that you develop the same approach toward the problems which will, undoubtedly, confront you in later life. Finally, you would do well, while still in a position to enjoy the privileges of university education, to develop the attitude of mind which strives, not only to take in, but also to lead out. Over three hundred years ago, Galileo observed, "You cannot teach a man anything: You can only help him to find it within himself." Of course, if it isn't there, it cannot be found.

And now I see utterly no reason why you cannot pass any examinations which may be set before you. If you can't, be assured the fault is yours. I have told you all that any teacher could, to help you. After all, you know, you must do some of this yourself.

Abstracts

C. L. ASZKANAZY, Editor

CONTRIBUTORS
C. L. ASZKANAZY, Editor
J. G. BELLAMY
W. B. WEISBROD

MEDICINE I

INFECTIOUS MONONUCLEOSIS. J. Meline: *New England Journ. Medicine,* Vol. 233, No. 24, Dec. 1945.

This disease never occurs in epidemic proportions. Usually the age group of 17-31 is attacked, the preponderance of patients being male.

Clinical manifestations: The symptomatology is protean in nature and non-specific. In the series reported by the author, most frequent was sore throat, varying from dryness to pain; malaise with generalized aches, fatigue and mild to severe frontal or generalized headache; feverishness and chills, accompanied by rigors if sore throat was present; nausea, vomiting, diarrhœa and abdominal pain; mild cold to severe cough.; dizziness and drowsiness; watering eyes and puffy eyelids.

Physical signs: Most of the patients showed inflammatory lesions of tonsils, pharynx or tongue with ulceration and abscess formation. The bacteriology was mixed or consisted predominantly of Vincent's spirillum or beta-hæmol. Strep. Moderate fever, 100-101° F., was present in all but 3 patients. In one case it reached 105° F. The average duration of the fever was 6 days. The cervical, axillary and inguinal lymphnodes showed moderate to marked enlargement in most cases. Splenomegaly was present in half the cases, the spleen being firm and non-tender. Jaundice, mild facial œdema, and erythema, ecchymoses or eruptions were occasional findings.

Blood Picture: On admission, the white count ranged from 4000-22000, with an average of 10600, but it increased as the disease progressed. There was a progressing relative neutropenia, with mononuclear increase of 60%, consisting of monocytes and normal and toxic lymphocytes. 90% of the patients gave a positive reaction in the heterophile antibody test.

Complications: These comprised chiefly transient albuminuria and peritonsillar and lingual abscess. Tachycardia, purulent otitis media, ruptured spleen, and broncho-pneumonia were present in one case each.

Treatment: There is no specific therapy. Sore throat is combatted with saline irrigation, pain with aspirin and codeine. Mouthwashing and gargling with Sod. perborate is helpful in Vincent's infection.

W. B. W.

MEDICINE II

Backpain as a Symptom of Carcinoma of the Body of the Pancreas. E. E. Kattwinkel: *Ann. Int. Med.,* Vol. 23, No. 6, Dec. 1945.

The diagnosis of carcinoma of the body of the pancreas is rarely made during life. Suspicion should be aroused by a history of backpain associated with indigestion aggravated by lying down and relieved by bending forwards. Other possibilities are excluded by physical examination, X-ray, and laboratory studies. A positive diagnosis can be made only by laparotomy and biopsy.

Laboratory work should include a determination of the serum lipase and amylase, which is raised in 40% of cases. Anæmia occurs late in the disease.

A case is reported in which the diagnosis was reached after exclusion of irritable colon, hypertrophic arthritis of the spine with radicular pain, duodenal ulcer, and pancreatic cyst.

W. B. W.

SURGERY I

Cutis Grafts. H. N. Harkins: *Annals of Surgery 122*(6) :996, Dec. 1945.

Methods of Obtaining Cutis:

(1) Loewe: Full thickness graft cut, epidermis scraped off with a scalpel.

(2) Rehn: Thiersch graft cut, cutis graft cut from exposed bed, Thiersch graft resutured.

(3) Rehn's Türflügel: In obese patients with incisional hernia, graft cut from part of abdominal wall to be discarded.

(4) Dermatome Flap: Like (2), but cut with a dermatome and epidermis left attached at one end.

(5) Scola: Cut full thickness (.04-.05″) graft, attached at ane end, shave off epidermis (.008″) which is resutured. All is done with a dermatome, so that here both sides of the graft are smooth. A sharp blade is needed and part of the graft is wasted due to the difficulty of detachment.

(6) Split-split: The skin is cut off, then the epidermis shaved off by resetting the blade on the drum of the dermatome. This is the best method.

Clinical Cases: 2 patients with epigastric, 9 with ventral incisional hernia were grafted. The duration of the herniæ was from 2-21 years and in 5 cases previous attempts at repair had been made. Methods 4, 5, and 6 were used. It was found to be unimportant whether the right or the fatty side faced the new bed. The thickness of the epidermis varied from 9-20/1000″, of the cutis from 20-31/1000″. All donor areas healed easily. A recurrence occurred only in 1 patient, 256 lbs., a diastasis on straining.

Experiments on Dogs: 25 cutis implants on 14 dogs were performed. Of 13 abdominal grafts, only 6 were without complications (pus, hair, disappearance of graft, etc.). Ligations of large arteries with grafts were unsuccessful, since they

either disappeared without producing occlusion or were converted into sebaceous material. Bridgings of 5 abdominal wall defects and 3 diaphragm. herniæ healed well, but showed hair microscopically.

Conclusions: Cutis grafts should be applied under tension and in hernial treatment fastened as supporting patch over the suture line. Full skin grafts are not suitable as buried patches for hernial repair since the epithelial elements can form cysts.

C. L. A.

SURGERY II

Intrathoracic Metallic Foreign Bodies. L. H. Garland: *Diseases of the Chest* *11* (*6*): 662, Nov.-Dec. 1945.

This article deals only with objects which have penetrated the chest. The diagnosis is based on history, presence of a scar, and clinical findings, all of which may be misleading, however. Most essential is röntgenography, the plates being taken both in the antero-posterior and lateral plane. The röntgenologist should report: (1) the number, size, shape, position and movement of opaque foreign bodies; (2) any changes in pulmonary and pleural structures, diaphragm, thoracic wall, mediastinum, heart and pericardium; (3) the presence of opaque foreign bodies in other parts of the body. If removal of the foreign body is necessary, the patient should be placed in operating position under the fluoroscope, and the position of the particles marked by a skin scratch or subcutaneous dye injection.

A report on 150 cases, mostly with shell and mortar fragments, is given. These men were seen 3-5 months after the injury. Most of the fragments were intrapulmonary, 20% with associated bone damage; 25% of the fragments were 1 cm. or more in diameter. Although the fragments had not been removed, 80% of the patients had no clinical symptoms at the time of the survey, and 10% had only slight ones; 75% had residual pleural, 15% pulmonary changes.

Late complications include recurrence of infection, developmental herniæ, more rarely migration of small slender objects, disintegration of the fragment (lead), erosion of blood-vessels, pleural irritation, and pressure on the phrenic nerve.

The author concludes from his survey that the vast majority of fragments does not need removal, irrespective of size. Other workers advise removal if the fragments are large, centrally placed, or cause complaints.

C. L. A.

SURGERY III

The Etiology and Treatment of Polycythemia Rubra Vera. P. W. Schafer: *Ann. of Surg.*, Vol. 122, No. 6, Dec. 1945.

Experimental Work: Using dilution methods, the average normal values of plasma vol., RBC mass, and total blood vol. were determined in 29 normal dogs: Following proprioceptor depressor neurotomy, 6 out of 13 animals developed polycythemia and all because hypertensive. Following extensive paravertebral sympathectomy, 4 out of 6 dogs developed a significant decrease in red cell mass and

total blood volume. 2 animals with chronic peripheral vasodilation which had resulted in transient minimal hypotension, were subjected to propioceptor depressor neurotomy. This resulted in moderate hypertension but no polycythemia occurred.

Clinical Case: A case of a 27-year-old man with intractable polycythemia vera is presented. Paravertebral sympathectomy was performed and 39 months after the operation, the man is asymptomatic and able to perform strenuous labour. The post-operative change in blood volume has been related to sympathetic efferent function. The theory is advanced that some cases of polycythemia in man are due to a construction of the presinusoidal blood vessels in the bone marrow. Extensive paravertebral sympathectomy is the treatment for such cases.

W. B. W.

OBSTETRICS AND GYNECOLOGY I

THE Rh FACTOR IN OBSTETRICS. H. F. Traut, J. Howard, L. Charnet, B. C. McIvor, S. P. Lucian: *Am. Journ. Obst. and Gynec.*, Vol. 50, No. 6, Dec. 1945.

In this article evidence is presented that the Rh factor, even when not recognizable clinically, is a causal factor in prematurity, certain abortions, enlarged liver and spleen, nucleated RBC in excess of 5%, and hydrocephalus, as well as in Hæmolytic Disease of the the Newborn or Erythroblastosis fœtalis.

The authors advocate:

(1) Avoidance of any analgesia or anæsthesia which might result in decreased 0_2 carrying capacity of the maternal blood during labour.

(2) Rh typing of all women who had transfusion reactions or who had repeated abortions due to fœtal anomalies of a suggestive nature; if such women are negative, their husbands should be similarly studied.

The fall in titre of Rh agglutinins in the mother may be an indication of increased absorption of antibodies by the fœtus; interruption of the pregnancy at this stage would then result in a reduced degree of damage to the infant.

Recommended treatment for Hæmolytic Disease of the Newborn includes:

(1) Prompt transfusion of the infant with Rh negative blood from some donor other than the mother.

(2) Red cell counts on the cord blood of all infants of Rh negative mothers, to be repated at 6 hour intervals on the infant's blood.

(3) Oxygen therapy.

(4) Vit. K is of no assistance in such cases.

J. G. B.

OBSTETRICS AND GYNECOLOGY II

THE SURGICAL CORRECTION OF CONGENITAL APLASIA OF THE VAGINA. N. F. Miller, J. R. Willson, J. Collins: *Am. Journ. Obst. and Gynec., 50(6):735,* Dec. 1945.

A short history of attempts at surgical correction is given, which include:

(1) Baldwin method: A loop of terminal ileum is carried downward through a newly dissected channel between bladder and rectum.

(2) Schubert method: The lower rectum, with the exclusion of the anal orifice and sphincter, is moved forward to function as vagina.

(3) Various methods which involve dissection of a channel between bladder and bowel and lining of this channel with skin derived from the labia or the thigh.

The authors consider either one of the following two procedures to be the best method:

(1) A channel is formed between the bladder and bowel and an obturator left in it until epithelialization has taken place by ingrowth from the vestibular epithelium.

(2) A channel is dissected into which is placed an obturator covered by a split-thickness skin graft.

Steps of procedure are given and a series of 17 cases is reported. In the majority of cases, the operation was successful, the best results being obtained with the skin graft technique. The normal skin from the thigh, transplanted into the artificially produced vaginal channel, appeared to assume the function of normal vaginal mucous membrane.

J. G. B.

PAEDIATRICS I

THE USE OF PENICILLIN IN THE TREATMENT OF ACUTE HAEMATOGENOUS OSTEOMYELITIS IN CHILDREN. E. W. Compere, W. J. Sehante, L. M. Cattell: *Ann. of Surg.*, Vol. 122, No. 6, Dec. 1945.

In the age group from 3 years to puberty, infection is secundary to a furuncle or boil of the skin which leads to bacteræmia and localization in one or more bones. If septicæmia supervenes, abscesses are formed throughout the body.

A series of 12 cases is reported, ranging in age from 3-12 years. The bones involved included: femur (4), tibia (5), humerus (1), ischium (1), cuneiform (1), calvarium (1), and patella with femur (1).

Penicillin was administered intramuscularly, 15,000-20,000 units every 3 hours day and night until the fever began to subside when 10,000 units were given. Treatment was discontinued after 5 fever-free days. Some patients also received sulphonamides. 1 child had a small sequestrum removed and 3 had abscesses drained after the acute phase of the disease was past. Other measures included maintenance of fluid balance by injections of glucose in saline or plasma; splints, plaster encasements and traction to support the diseased bone and prevent contractions.

There were no deaths in this series nor any extensive areas of chronic osteomyelitis. Multiple foci in bones disappeared as soon as treatment was begun. Penicillin was effective where sulphonamides failed.

W. B. W.

PUBLIC HEALTH I

EDUCATING FOOD HANDLERS. M. J. Harper: *Can. Journ. Pub. Health 36 (12)*: 465, Dec. 1945.

A report is given on the measures taken in the Prince Rupert Health Unit—18,000 square miles—to secure sanitary conditions in restaurant and cafés

From 1942 to 44 regular inspection tours were held. At the beginning of 1945, 75% of the establishments were approved and were given window cards to show it. Food handlers received a course during 2 evenings, including lectures and demonstrations on bacteria, proper technique, and how food handlers can help to control disease. Notes of instruction (12 pages) were distributed on which a true and false test of 32 questions was held. A pass mark of 65% was required to obtain an accredited food handler's certificate. Certificates and window cards could be revoked when flagrant breaks in technique of the individual or restaurant occurred and the Health Unit approved only premises where 75% of the personnel held certificates. The Prince Rupert Restaurant and Café Owner's Association resolved that after 3 months, members must hire only certified cooks, kitchen help, and waitresses.

C. L. A.

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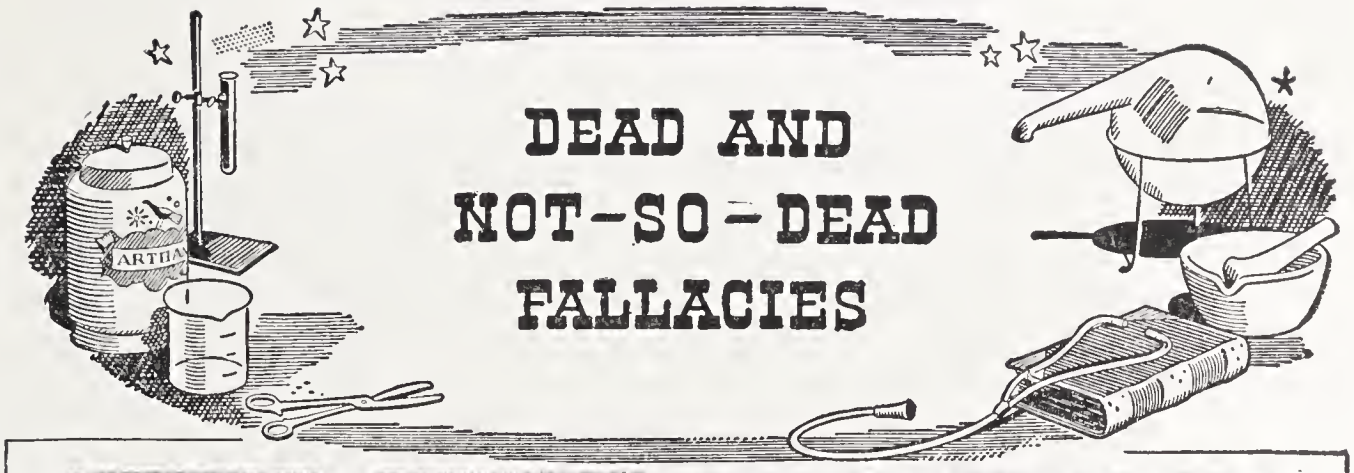

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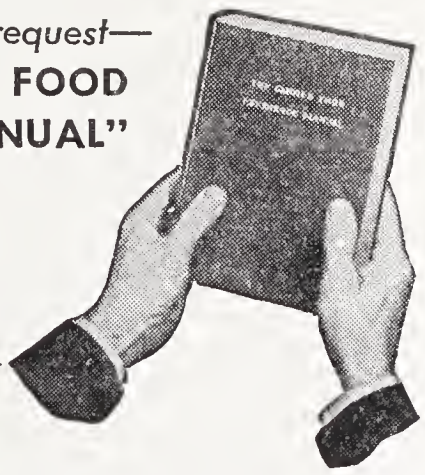

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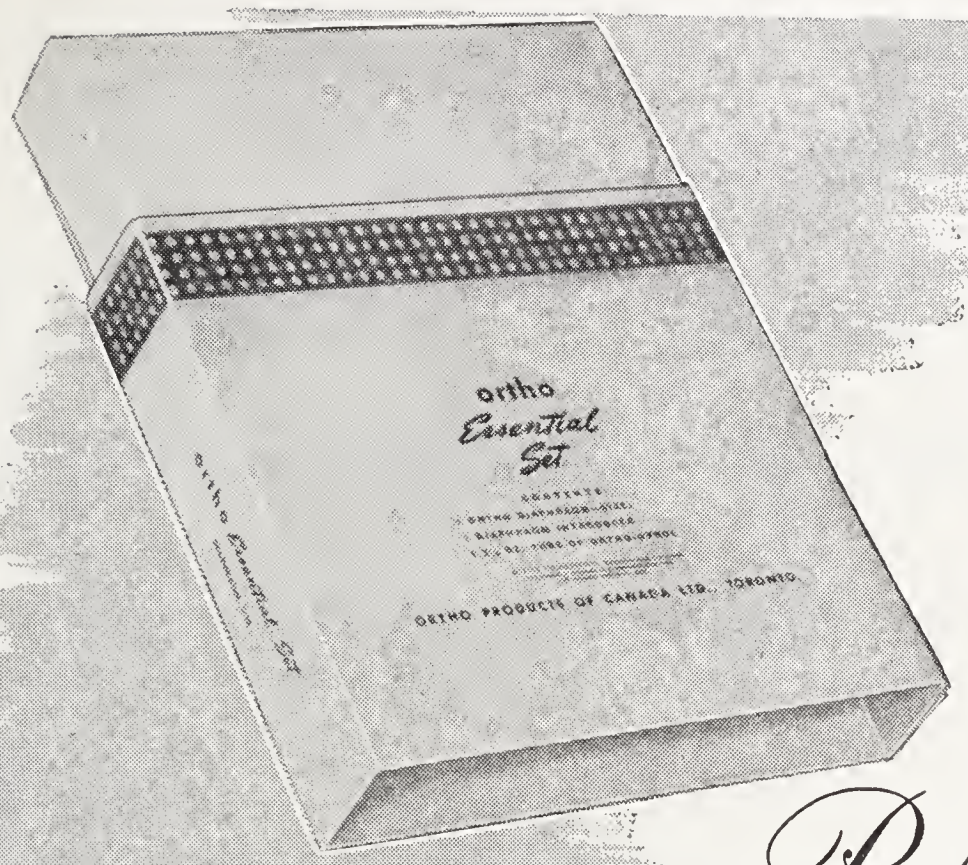

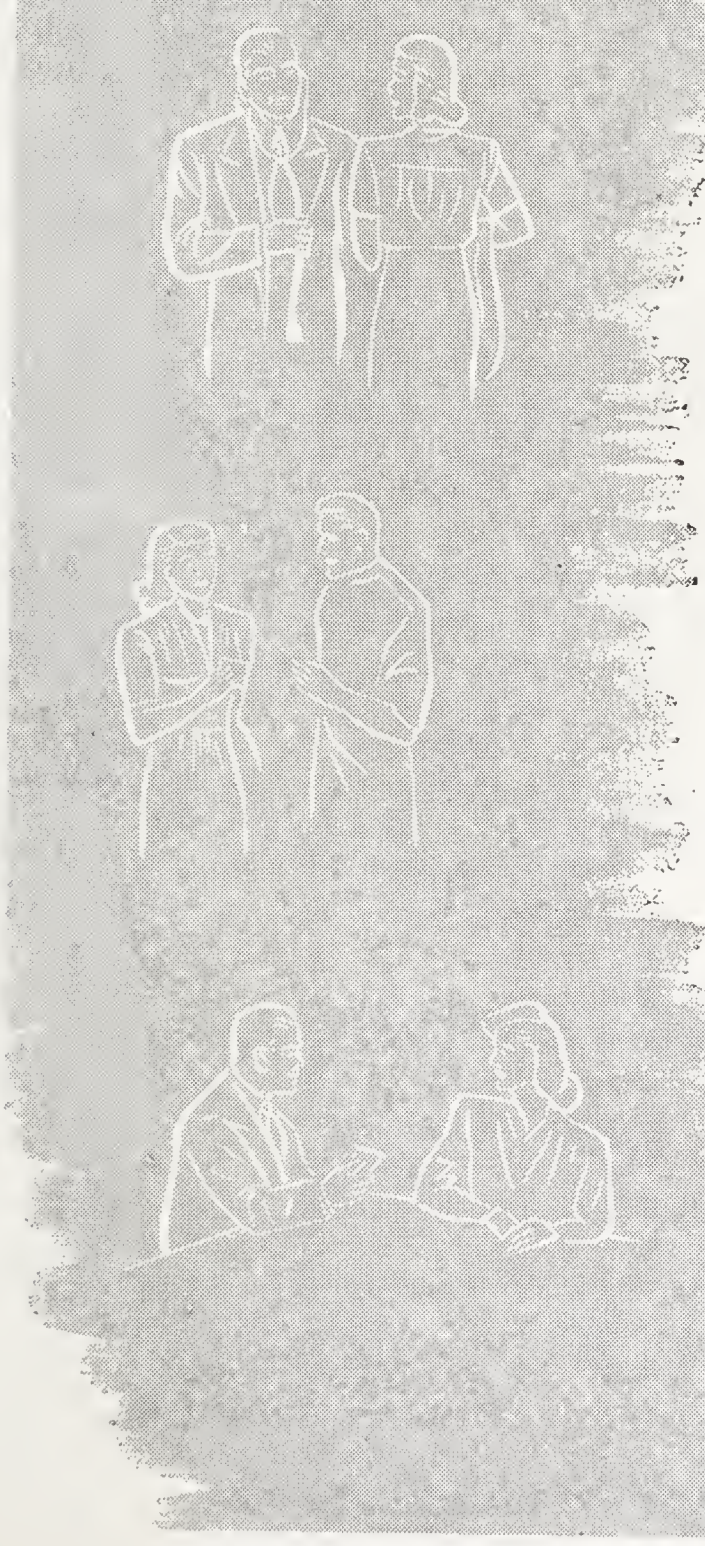

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*R. H. Follis, D. Jackson, M. M. Eliot, and E. A. Park: Prevalence of rickets in children between two and fourteen years of age, Am. J. Dis. Child. 66:1-11, July 1943.

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CORRESPONDENCE

A few years ago the University of Toronto Medical Journal was good enough to publish a short essay of mine on Medical Argot. In it I protested against the spelling of the principal alkaloid of Opium as "Morphia." One of my colleagues objected to my attitude by saying "I prefer the British to the American usage." That absurd idea may account for my finding a goodly number of students writing 'morphia' for 'morphine' in their answers to a certain question at the recent final examination.

The facts are that the Pharmaceutical Commission of the General Medical Council of Great Britain in the year 1900 ruled that the names of *all* alkaloids should end in—'ine' (English) and—'ina' (Latin). The ruling was adopted in the United States and so became international. Previous to 1900 the rule of nomenclature was that the names of *all* alkaloids should end in—'ia', genitive—'ae'. The teachers who use the old form would resent being dated as having qualified so long ago!

In the past ten years I have indulged myself in accusing editors of certain British Journals and authors of popular text books of being very old men. The results so far have been promises from Editors of the British Journal of Surgery, the British Journal of Anaesthiology and the Lancet that morphine as well as codeine should be spelled according to rule and not according to a tradition that is simply a bad habit. The Oxford Dictionary refused to omit 'morphia' as an alternative spelling for morphine on the ground that it exists in popular use. That does not excuse the anachronism.

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